ditions co-existed. On the basis of the above observations it would be expected that the behaviour of the blood volume and hæmatocrit would be influenced in opposite directions by these two clinical states and the final result would be due to the balance between them. In the 4 cases where both shock and cardiac failure were present, the blood volume fell in 2 with a concomitant rise in hæmatocrit. In the other two cases, the blood volume remained unchanged, the cell volume remaining unchanged in one of these and varying in the other. In the one case showing shock unaccompanied by clinical evidence of cardiac failure the blood volume rose while the hæmatocrit reading fell. In the one case where there was cardiac failure and no shock the blood volume again rose somewhat but the hæmatocrit remained unchanged. This patient showed very marked right-sided cardiac failure, the rise in blood volume coinciding with a deterioration in his clinical state. In 2 cases where no shock or failure existed clinically the blood volume rose while the hæmatocrit values fell, indicating that the conditions found in shock were present but were not clinically manifest. In 2 cases also without clinical evidence of shock or failure the blood volume fell. In one of these the hæmatocrit values fell and in the other they varied. These results may be attributed either to subclinical cardiac failure or the effect of bed rest alone (Taylor and Erickson[14]).

SUMMARY AND CONCLUSIONS

1. No constant changes in blood coagulability as measured by the Waugh-Ruddick test, plasma prothrombin time or coagulation time were noted in 31 cases of acute coronary thrombosis. No constant changes in the Waugh-Ruddick test resulted from prolonged bed rest in the cases studied.

2. No constant variation was observed in plasma protein levels during convalescence.

3. A high percentage of patients suffering from acute coronary thrombosis have prolonged circulation times, due either to shock or to myocardial weakness, or to a combination of these factors.

4. The results of blood volume studies in these cases support the view widely held that the values vary with the presence or absence of cardiac failure and/or clinical shock.

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STUDIES IN PROGRESSIVE LIPODYSTROPHY*

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IN 1885, Weir Mitchell described to the Philadelphia Neurological Society a "Singular Case of Absence of Adipose Matter" in the upper half of the body of a twelve year old girl. This curious condition followed an attack of severe cold with cough and expectoration which lasted for three months at the age of five years.[1] Ten years later a similar patient was observed by Osler: he noted the sharp contrast between the extremely emaciated face and trunk and fairly plump lower part of the body in a ten year old girl. The wasting had begun at five years of age. This case did not appear in the literature at that time but was subsequently reported by Weber.[2] The disease was not recognized as a clinical entity, however, until Barraquer in Barcelona[3] and a year later, Campbell in England[4] published their studies in this curious condition. Other cases have been reported[5, 6] since that time, but the first detailed study of this disease was made by Simons[7] who named it lipodystrophy progressiva. It is also known as Barraquer-Simons disease, lipodystrophy progressiva superior, and craniothoraco lipodystrophy.

By 1940 about one hundred cases were reported from different parts of the world[10] but

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many more must have remained unrecognized since the disease is self-arresting.

Onset and course.—Progressive lipodystrophy is more common among females and is characterized by gradual disappearance of subcutaneous fat usually first from the face, then affecting the neck, shoulders, arms and trunk. In some cases (more common among men) the atrophy is confined to the face and neck alone.[11] In females there frequently is a considerable increase of subcutaneous fat in the lower half of the body. In a few cases the conspicuous enlargement of the buttocks and thighs was reported to precede the loss of fat from the upper part of the body. Thus the disease may combine both the *atrophic* and *hypertrophic* features at once.

The onset of this condition is usually quite insidious. The loss of subcutaneous tissue becomes apparent as a bilateral symmetrical thinning of the face occurring before puberty, frequently in the first decade of life. No obvious disturbances of glandular activity are present. Some authors reported the presence of menstrual irregularity and even sterility in females; however, many of the female patients having progressive lipodystrophy had married and given birth to normal children. Usually the loss of subcutaneous fat spreads from the neck to the shoulders, arms and then trunk. This feature is responsible for the term "progressive" but the progress of the disease ordinarily comes to a spontaneous arrest when the subcutaneous fat has disappeared from the upper part of the body.

Although the patient may have some vague complaints during the "active" period associated with the loss of subcutaneous fat, no specific symptoms have been found and all systems are apparently normal. By the time the progressive feature of the disease has run its course, thin skin alone covers the bony framework and the muscles. The typical appearance of such an individual is characterized by deep-set eyes, prominent malar bones, hollowed temples and cheeks and the whole face is deeply furrowed. The neck is usually thin and stringy and the thorax is bony, with prominent clavicles, scapulæ and vertebræ. The breasts in the female are usually small and pendulous, the muscles are clearly defined and appear to be over-developed, the abdomen is thin and scaphoid. The buttocks on the other hand, are usually obese and so are the lateral aspects of the thighs and lower legs. The ankles are seldom involved.

Laboratory investigation.—Biochemical investigations yield little positive contributory information and in some cases conflicting findings have been reported. Several workers reported some difficulty in the deposition and, to a lesser extent, in the oxidation of fat.[10] The absorption of fat is apparently normal. The levels of blood cholesterol vary from 142 to 330 mgm. %.[10] Those of serum calcium, serum phosphorus,[13] blood sugar,[8, 14] blood urea, nitrogen, uric acid, non-protein nitrogen, creatine and creatinine[10] are all in normal range. The number of cases in whom extensive biochemical investigations have been carried out is not very great, but so far, no specific laboratory findings diagnostic of progressive lipodystrophy have been found. In one of our patients in whom certain endocrine functions were investigated, no abnormalities were found.

Pathology.—Although over 100 cases of progressive lipodystrophy have been reported, only a few of the investigators have presented biopsies of skin and subcutaneous tissue from affected regions of the body.[7, 8, 12, 16, 17] Complete autopsies were performed in only two cases, both under 15 years of age,[15, 18] who died from intercurrent infections not associated with lipodystrophy. In spite of the numerous reports of various pathological conditions associated with progressive lipodystrophy, we agree with Harris and Reiser that there are no constant pathological changes except for the lack of subcutaneous fat in the affected areas of the body. The skin and muscle appear normal, both clinically and histologically. On examination, no impairment has been found either in the function of the muscles or in the response to electrical stimulation of either the muscles or nerves. Disturbances of sensory sensation, reflex or vasomotor function have not been reported.[19]

Etiology.—Numerous attempts have been made to incriminate various factors as the cause of progressive lipodystrophy but the etiology of this condition remains obscure. There appears to be no hereditary or racial predilection although these have been suggested by some authors.[10, 11] Others[20] have considered that the condition is due to local congenital anomalies, which is unlikely. Brain[25] attributed the cause of lipodystrophy to disturbance of autonomic innervation possibly due to infection. Some

workers considered basal meningitis or hydrocephalus as one of the underlying causes.[22] Others presuppose the presence of some defect in the diencephalic centre, possibly congenital in origin, which manifests itself only when other difficulties arise such as commencement of the menstrual cycle, gestation, menopause, or trauma (due to infection etc.).[14, 22, 23]

Infections.—In many cases the onset of progressive lipodystrophy was reported to follow some antecedent infectious disease such as measles, pneumonia, rheumatic fever, mastoiditis etc., but the significance of such infectious diseases in the pathogenesis of this condition is not clear. Many authors consider them merely intercurrent or antecedent diseases.

Endocrine dysfunction.—Some fat dystrophies are known to be associated with endocrine dysfunctions: Frohlich's syndrome, Dercum's disease,[39] diabetes, hyperthyroidism, hypothyroidism, Addison's disease, etc. Almost every endocrine system of the body has been considered as the etiological factor underlying progressive lipodystrophy. The pituitary,[10] thyroid,[11, 12, 21] gonads,[8, 9, 22] pineal gland,[24] and hypothalamus have been incriminated in the etiology but there is no concrete evidence to support this nor for the existence of a centre of fat distribution[27] to account for subcutaneous fat changes peculiar to progressive lipodystrophy.

Neurogenic and trophic changes.—In several cases the onset of lipodystrophy followed an attack of encephalitis or menginitis. Moreover, some authors considered that there is a definite increase in the incidence of lipodystrophy following acute infections which may affect the central nervous system. Possibly because alterations of fat metabolism have been asociated with various cerebral changes (dystrophia adiposogenitalis), obesity secondary to basilar meningitis, hydrocephalus of the third ventricle, fractures of the base of the skull,[17] a neurogenic etiology has been proposed by some authors.[3, 14, 17, 22, 23] Trophic disturbances have been found in association with progressive lipodystrophy in some patients.[14, 22 to 24, 26] At present the available clinico-pathological findings do not lend support to any of the etiological hypotheses proposed and these must remain simply as theoretical assumptions. The occurrence of somatic, trophic, endocrine and functional aberrations in association with progressive lipodystrophy may be purely coincidental, and not in any way contributory to the causation of this condition.

TREATMENT

Since the etiology of progressive lipodystrophy is unknown, there cannot be any form of treatment which has a specific clinical basis for its application. The various therapeutic measures tried, comprised administration of thyroid, pituitary and ovarian extracts, massage, electricity, hydrotherapy, bed rest, diet, etc. Overfeeding produces an increase of fat deposits in the lower extremities but no change in the atrophied regions although some authors disputed this.[28, 29] There appears to be no specific general treatment of progressive lipodystrophy. Fortunately the condition is self-limiting[30] and progresses no further when all fat has been lost from the upper part of the body. However, the sequelæ of the disease due to altered appearance usually generate a number of problems which have social, economic and psychiatric implications. The cadaverous appearance of the face which is so characteristic of patients afflicted with progressive lipodystrophy causes them to withdraw from society. Such patients usually avoid crowds, have few friends and do not want to be seen outside their homes in the daylight. It is difficult for them to find employment because both the employer and employees consider that their appearance suggests the presence of some serious disease. Before long, several symptoms such as restlessness, insomnia, phobias, inferiority complexes and other complaints, appear.

Generally, however, in spite of their "sickly" look, these patients enjoy good health and are able to work as well as the normal individual. Surgical reconstruction of the obvious facial defect is sufficient to restore the patient's self-confidence so that he is able to enjoy normal relationships both socially and economically.

Local therapy.—Transient improvement has been obtained by the injection of human fat and sheep's suet[6] and of paraffin[16] subcutaneously. These methods are now in disrepute and for the past two decades autogenous grafts have been used to correct the depressed areas on the face. Moskovitz[31] employed strips of fascia lata and fat. Straatsma[32] described the use of transplants of dermis and fat for small defects. Cotton[33] inserted finely cut gluteal fat. Other experimental studies have shown that this latter method leads to greater absorption than when a single large piece of fat is used.[34] In order to

avoid shrinkage of the transplant Eitner[35] used island flaps of skin and fat from the temporal region. For the treatment of hemiatrophy of the face, Byars[36] has described the use of various methods; fascia and dermis with or without fat, or decorticated pedicle flaps inserted through submandibular incisions. Kazanjian[37] found that the best results in hemiatrophy were obtained by the insertion of blocks of dermis, fat and fascia. The graft may be cut into small cubes or applied in one large piece. During the past few years, the insertion of alloplastic grafts such as tantalum sheet and wool, or acrylic implants in depressed areas of the face has enjoyed quite a vogue. Most of these were defects due to trauma. We believe, however, that in due time a final appraisal of these cases will be made and the advantages of autoplastic grafts will result in their use exclusively.

Three patients with progressive lipodystrophy have been treated by us and the most successful result was obtained by the use of large dermal grafts with a small amount of fat. In one case in whom a block of fascia and fat was used, there was marked absorption. However, from the experience of these cases it would appear that dermis grafts alone offer the most permanent æsthetic results because dermis is absorbed more slowly and much less completely than fat. Some of our biopsies taken two years after the insertion of dermis and fat grafts show identifiable elements of the dermis remaining although there was considerable deposition of fibrous tissue in the immediate vicinity of the graft (Fig. 3, D). The possibility of the formation of epidermal cysts in the graft has been completely allayed by the excellent experimental work of Peer and Paddock.[38]

OPERATIVE PROCEDURE

General considerations.—It is hardly necessary to mention that extreme gentleness should be used in obtaining and in trimming the graft, so that tissue damage from trauma and subsequent necrosis may be avoided. In no instance has perforation into the oral cavity occurred while the skin flaps were being dissected from the underlying muscle. However, the possibility of this renders it advisable to separate the skin flaps from the face before removing the dermal grafts so that in the event of perforation reconstruction may be postponed. In our first patient, we used folded sandwiches of fascia lata and fat. In the subsequent two patients, dermis and fat grafts were inserted, and the amount of shrinkage was much reduced. In the future it is planned to insert only sheets of dermis, built up in layers to conform to the size of the defect.

Determination of size of graft.—One procedure for obtaining the dimensions of the graft required, is to take an impression of the patient's face and pour a plaster model. The normal contour of the face is then restored with wax and the dermis graft may then be shaped to correspond in size to the wax pattern. An alternative and simpler method is to fashion pads of sponge rubber directly on the patient's face which fill the defects in contour. These may be autoclaved and when the grafts are removed they can be trimmed and built up so that they reduplicate the rubber models almost exactly.

Donor area.—In all three cases the measured grafts were taken from the upper lateral aspect of the thigh. A dermatome skin graft 0.008 inches thick was removed after which two pieces of dermis of predetermined size were excised. The epidermal graft was resutured in the defect and a routine pressure dressing was then applied.

Insertion of the grafts.—We have used a part of the usual face lift incision for elevation of the skin over the cheeks. This extends from the superior junction of the ear with the scalp into the external auditory meatus behind the tragus, and then emerges to extend down to the junction of the lobule with the skin of the cheek. Thus a most inconspicuous scar is the result. The skin of the cheek is dissected with sharp scissors in a plane just beneath the dermis. This is most important, if free bleeding is to be avoided. Should this be encountered, the cavity is packed with warm saline dressings and firm pressure applied. If this does not control oozing, gauze sponges saturated in a 1:1,000 solution of thrombin may be inserted and external pressure maintained by the assistant while the opposite cheek is being prepared. When bleeding has been controlled, the transplant is inserted in a single large block. Sutures of No. 38 stainless steel wire are placed in mattress fashion at the four corners of the graft and long straight needles are threaded on each end. The needles are passed through the skin of the face at predetermined points, so that the graft will be kept at normal tension and correct position in the cavity. The wire sutures are then tied over small rolls of xeroform gauze.

In all instances, the dermal side of the graft has been applied to the under surface of the skin

of the cheek in anticipation that a firmer degree of fibrosis would unite the two and thus prevent contraction of the implants. As a precaution against infection, a few cubic centimetres of a solution of penicillin containing 25,000 units per c.c., is injected into the cavity before the skin incisions are closed with fine sutures. Because the formation of a hæmatoma is so detrimental to the survival of large implants, a strand of silkworm gut is inserted at the inferior end of the incision to act as a drain for 48 hours.

Postoperative care.—Before application of the dressing, the patient's teeth are wired together to aid in immobilization of the operative site. A firm pressure dressing is then applied evenly over the face. A soupy diet rich in calories and vitamins is then given. At the end of two weeks, the wires are removed. The pressure dressing is retained for a week or two longer.

Complications.—The most serious complication which may occur is the development of infection. Preventive measures are to be stressed but should it occur, dependent drainage may be obtained by opening the inferior portion of the suture line. Penicillin should be employed generally as well as locally. If a hæmatoma forms in spite of the usual precautions, it should be evacuated by aspiration or re-opening part of the suture line. Failure to do so, if the collection is large, will interfere with prompt revascularization of the grafted tissue which will subsequently atrophy even if infection does not develop. A third reason for failure is the insertion of the graft without mattress sutures to retain the tissue under normal tension during the process of healing. Movement of the facial muscles and jaws will tend to roll the flap into a thick lump. If the cosmetic appearance is unsatisfactory as a result of any of these complications a new graft of appropriate size may be inserted to fill in the defect. This should not be attempted until the reaction from the initial operation has completely subsided. With care the desired result can be achieved by one operation.

The following protocols of the three cases of progressive lipodystrophy which we treated illustrate the application of the principles outlined above.

CASE 1

Miss E.E., aged 25 years, was admitted to the Plastic Surgery Service complaining of increasing wasting of the face, chest and abdomen (Fig. 1, B). The loss of fat began in her "early teens" and eventually a relative increase in the size of her hips and thighs occurred. At the age of twenty years the patient suffered from an attack of quinsy, complicated by severe septic throat and mastoiditis. Following this illness the thinness of the upper part of the body became more pronounced, but there were no other signs or symptoms. The functional inquiry was entirely non-contributory with the exception of the presence of some dysmenorrhœa in the few months preceding admission to the hospital.

Physical examination revealed extreme loss of fat in the face (Fig. 1, B), chest, and upper abdomen. The hips were well developed with normal distribution of fat. No muscular atrophy was present. Examination of all systems produced no evidence of abnormality. Laboratory investigations revealed no abnormal findings.

Operation.—Under spinal anæsthesia fat-and-fascial grafts were removed from the right and left thighs and inserted, under local anæsthetic (novocaine 2%), into subcutaneous pockets formed by raising the skin of the right and left cheeks through modified face lift incisions. All sutures were removed on the 10th postoperative day. A hæmatoma had formed in the right cheek and aspiration was necessary on several occasions, however, no infection developed. The patient was discharged three weeks following operation. She was re-examined at intervals for six years and the marked overfilling of her cheeks which was carried out intentionally to allow for subsequent shrinkage, gradually diminished until the appearance shown in Fig. 1, D was obtained. Her social readjustment and mental outlook have improved steadily and at present she is well adjusted in spite of a small irregularity of the left cheek which she does not consider of sufficient importance to have corrected.

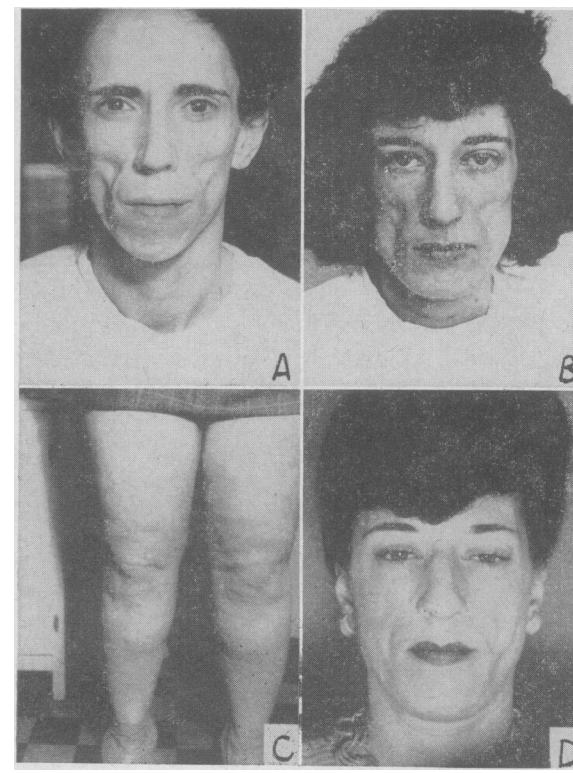

Fig. 1. — A.—Case 2, preoperative showing the cadaverous appearance suggestive of the presence of some grave illness. B.—Case 1, preoperative. Note the sunken cheeks and relative prominence of the orbicularis oris muscle. C.—The very obese lower extremities of Case 2. Such disproportion is not infrequently found in this disease. D.—Case 1 taken six years after the corrective operation.

CASE 2

Mrs. G.S., 37 years old, was admitted to the Plastic Surgery Service with the complaint of extreme thinness of the face and thorax for the past 20 years (Fig. 1, A) and dizziness for about three years prior to admission. The onset of the "thinness" appeared to have followed an attack of rheumatic fever at the age of 16 years. Every attempt to gain weight by increasing her food intake resulted in an increase in the deposition of fat in the lower part of the body, particularly the hips and legs. The face, neck, chest, shoulder and arms remained very thin. For the past three years she had been complaining of headaches and dizziness. The latter symptoms were most frequently noticed when she was in crowded places. She claimed that these symptoms were considerably improved by a course of liver and iron therapy.

Physical examination revealed gross absence of subcutaneous fat on the head, arms, thorax and upper abdomen. Her muscular development was excellent. On the other hand her hips and legs were abnormally obese, (Fig. 1, C). The blood pressure was within normal limits and all laboratory investigations were essentially negative. An endocrinological study was performed by Dr. J. S. L. Browne. The pregnandial excretion was found to be normal, indicating normal ovarian function.

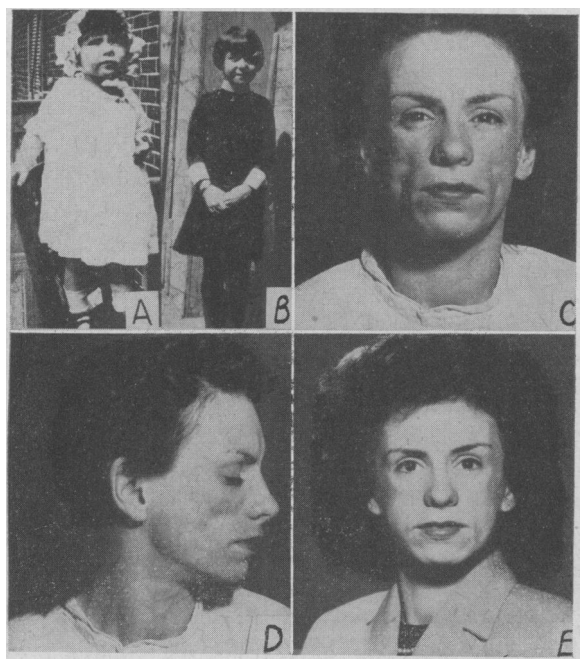

Fig. 2.—A. and B.—Case 3, showing the normal appearance at the ages of two and six years respectively. C. and D.—Her appearance at 27, just preoperatively. E.—Four months postoperatively.

Operation.—Under spinal anæsthesia, dermis and fat grafts were removed from the left thigh and inserted in the usual manner under the elevated skin flaps of the cheeks. Here again a small hæmatoma formed in the right cheek and was expressed a few days postoperatively, after removing a few sutures at the lower part of the incision. No infection occurred. Considerable shrinkage took place during the course of a year and she will require supplementary grafts to overcome the depression in the region of the naso-labial folds where the grafts were displaced backwards.

CASE 3

Miss M.C., 27 years old, was admitted to the Plastic Surgery Service with the complaint of wasting of skin over the face , arms and upper trunk, which started at the age of eleven and gradually progressed to her present appearance (Fig. 2, C and D). She was perfectly well until eleven years of age and had only measles and whooping cough at the age of 6 years (Fig. 2, B). Since then she has been in perfectly good health and started work at the age of 20 years. Because of her appearance she formed few friends, avoided crowds, and would not go out during the day except when going to work. She presented herself and inquired if something could be done for her appearance so that her social and business life would be rendered more agreeable.

Physical examination revealed a complete absence of external fat on the head, arms, thorax and upper abdomen. The breasts were well developed but only glandular tissue could be palpated. Her musculature development, due to lack of fat, appeared to be even more prominent than normal. Her hips and legs were of normal feminine configuration. The usual laboratory investigations were entirely within normal limits. Biopsies of the skin of the face and arm revealed a complete absence of subcutaneous fat on histological examination (Fig. 3, A and B). On the other hand, the histological examination of the skin and subcutaneous tissue of the thigh was completely normal grossly and histologically.

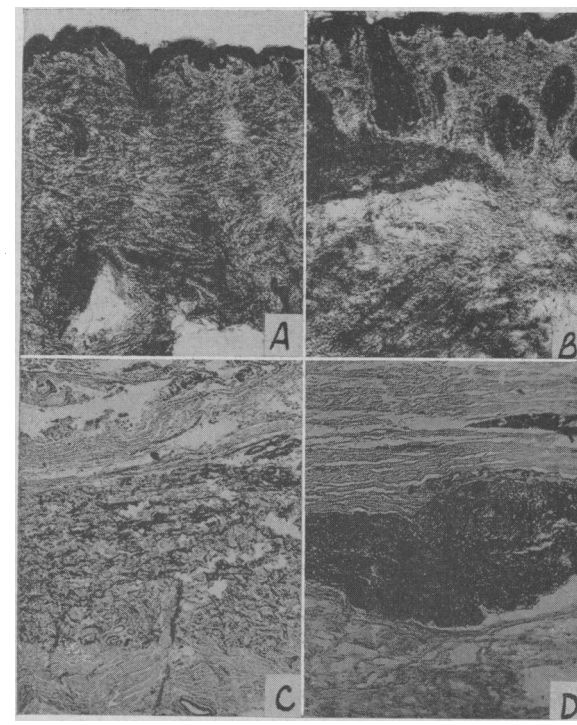

Fig. 3.—A.—Biopsy of the skin and subcutaneous tissue of the cheek showing the absence of subcutaneous fat (Case 3). Scharlach R fat stain. Magnification x 40. B.—Biopsy of the skin and subcutaneous tissue of the arm showing the complete absence of subcutaneous fat which would be represented by solid black in the section (Case 3). Scharlach R fat stain. Magnification x 40. C.—Biopsy of the dermal graft taken from Case 3 four months after implantation under the skin flap of the cheeks (Case 3). Verhoeff's elastic tissue stain. Magnification x 40. D.—Biopsy of the dermal graft taken two years after implantation. The dark-staining mass in the centre is made up of the persisting elastic tissue fibres. Verhoeff's elastic tissue stain. Magnification x 100.

Operation.—Under general anæsthesia, dermis grafts containing a minimal amount of fat were removed from the right thigh and inserted in the customary manner under the elevated skin flaps of the cheeks. Continuous

pressure was applied to the right cheek while the operation on the left cheek was carried out. The healing was prompt and uneventful. A biopsy was obtained from the dermis and fat graft in the cheek four months following operation and histological examination revealed no trace of epithelial elements, some scar replacement of the dermis, and absence of fat (Fig. 3, C).

SUMMARY

1. Progressive lipodystrophy is a disease which is more common in females. It usually starts in the first or second decade of life and is limited to the upper half of the body.

2. Laboratory investigations have revealed no diagnostic findings which are specific for this condition. Absence of the subcutaneous fat in the affected regions of the body is the only constant histopathological finding.

3. Certain infectious diseases, endocrine dysfunctions and neurogenic disturbances have been proposed by various investigators as possible etiological factors in this disease but no conclusive proof of any of these claims has been produced.

4. Although this disease is self-limiting it leaves the person with a "consumptive look" which makes it difficult for him to obtain work and drastically curtails his social activities. The development of inferiority complexes and of other psychological disturbances is almost inevitable.

5. Various types of general therapy have been unsuccessful so far. Local surgical therapy designed to fill in the sunken cheeks is a definite step toward reconstruction of the most obvious defect. Dermis or dermis and fat grafts are the most satisfactory autoplastic substances which may be used. Hæmostasis must be thorough; the graft should be sutured under tension and the jaw completely immobilized to permit fixation of the graft in correct position and to promote more rapid healing.

6. Sheets of dermis built up in layers and fashioned to conform to the shape of the defect appear to give the most satisfactory results. The techniques of operation and the possible complications are discussed.

7. Detailed studies of three cases of this uncommon condition have been presented and the method of treatment described.

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THE TREATMENT OF CARCINOMA OF THE CERVIX UTERI*

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THE purpose of this paper is to discuss certain points of the natural history of the disease, to review some of the published literature on the treatment of the disease and to present the results obtained at the British Columbia Cancer Institute by radiation therapy.

Carcinoma of the cervix is the second most common form of cancer in women. The disease occurs commonly in women between the ages of 40 and 50. It can occur before puberty, but before the age of 30 and after 60, the disease is rare. The disease occurs at an earlier age than carcinoma of the body of the uterus. There is no association between the number of pregnancies and the development of carcinoma of the cervix. The most common symptoms are vaginal hæmorrhage and discharge unrelated to the menstrual periods.

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